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Inhibition of tumor cell adhesion to lymph nodes by laminin-related peptide and neuraminidase.

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BACKGROUND. Adhesion to lymph nodes, rather than growth stimulation, accounted for preferential colonization of lymph nodes by a metastatic B16 melanoma. We investigated these adhesive interactions. METHODS. Four classes of molecules were tested for inhibition of melanoma adhesion to cryostat sections of lymph node. RESULTS. Calcium chelators ethylenediaminetetraacetic acid and ethyleneglycol-bis-(beta-aminoethylether)-N,N,N',N'-tetra ace tic acid completely inhibited adhesion (50% adhesion, half-maximal inhibition, at 1 to 3 mmol/L). Cytochalasin B, which impairs contractile microfilaments, inhibited adhesion (60% adhesion at .001 mmol/L, 28% at .01 mmol/L). Colchicine, which disaggregates microtubules, had a similar effect (20% at .01 mmol/L, lowest dose tested). Trypsin slightly increased adhesion (125% adhesion at 10 micrograms/ml). Neuraminidase, which removed sialic acid residues, inhibited it (50% adhesion at 5 micrograms/ml). Gly-arg-gly-asp-ser, a peptide with a cell binding sequence of fibronectin, did not consistently inhibit adhesion (69% adhesion at 0.1 mg/ml, 83% adhesion at 1 mg/ml) or substantially differ from gly-arg-gly-glu-ser-pro (59% adhesion at 0.1 mg/ml, 90% adhesion at 1 mg/ml). In contrast, a peptide with a cell binding region of laminin (tyr-ile-gly-ser-arg) inhibited adhesion (50% adhesion at .05 mg/ml). CONCLUSIONS. Tumor cell-lymph node adhesion is a calcium-dependent process, requiring a functional cytoskeleton, that is mediated by both sialic acid moieties and trypsin-resistant, laminin-related, adhesion molecules.

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